Smoking and Suicidal Behaviors in the National Comorbidity Survey: Replication

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Abstract: Controversy exists about the role of mental disorders in the consistently documented association between smoking and suicidal behavior. This controversy is addressed here with data from the nationally representative National Comorbidity Survey-Replication (NCS-R). Assessments were made of 12-month smoking, suicidal behaviors (ideation, plans, attempts), and DSM-IV disorders (anxiety, mood, impulse-control, and substance use disorders). Statistically significant odds ratios (2.9–3.1) were found between 12-month smoking and 12-month suicidal behaviors. However, the associations of smoking with the outcomes became insignificant with controls for DSM-IV mental disorders. Although clear adjudication among contending hypotheses about causal mechanisms cannot be made from the cross-sectional NCS-R data, the results make it clear that future research on smoking and suicidal behaviors should focus more centrally than previous research on mental disorders either as common causes, markers, or mediators.

Key Words: Epidemiology, mental health, mental disorders, nicotine, smoking, suicidal ideation, suicide plans, suicide attempts.

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A number of large prospective epidemiological studies have documented positive associations between smoking and subsequent suicide (Angst and Clayton, 1998; Hemmingsson and Kriebel, 2003; Leistikow et al., 2000; Miller et al., 2000a,b; Tanskanen et al., 2000). Smaller studies, most of them cross-sectional (Hallfors et al., 2004; Hintikka et al., 2001; Wu et al., 2004) but some prospective (Breslau et al., 2005; Hintikka et al., 2001), have also documented strong associations between smoking and suicide attempts. A lively debate has ensued as to whether these associations are causal or due to unmeasured variables that cause both smoking and suicidal behaviors (Miller et al., 2000a,b; Smith and Phillips, 2001; Smith et al., 1992). As smoking and nicotine dependence are known to be correlated with mental disorders (Breslau et al., 2004a,b; Grant et al., 2004) and, at least among adolescents, with many other psychosocial risk factors for suicide (King et al., 2001; Merrill et al., 1999), one side of this debate has argued that smoking is a proxy for these other unmeasured causes rather than itself a cause of suicidal behaviors (Smith and Phillips, 2001). The other side has argued that smoking might have

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causal effects on suicidal behaviors through such processes as: (a) effects of smoking on chronic physical illness that, in turn, lead to suicidal behaviors (Leistikow et al., 2000); (b) effects of smoking on depression or other mental disorders through various biological pathways (Balfour, 2002; Malone et al., 2003) that lead to suicidal behaviors; (c) effects of nicotine withdrawal on mental disorders (Covey et al., 1997) that cause suicidal behaviors; and (d) effects of early smoking as a sensitizing "gateway" that predisposes to use of more deviant drugs (Kelly and Rowan, 2004) and to involvement in more deviant behaviors (Amos et al., 2004) that, in turn, cause suicidal behaviors.

One way to study common causes is to investigate specificity of effects. For example, a widely cited study by Smith et al. (1992) showed that smoking predicts homicide as strongly as it predicts suicide even though, according to the authors, the plausible biological pathways linking smoking to suicide do not apply to the link between smoking and homicide. Smith et al. concluded from this result that the association between smoking and suicide is due to unmeasured common causes rather than to causal effects of smoking. A limitation of this argument is that it hinges on untested assumptions about causal processes involving the putatively irrelevant outcome, leaving the argument open to criticism based on evidence about causal pathways linking the predictor to the latter outcome. Research documenting effects of smoking on amino acids that might be biological determinants of impulsive aggression (Hibbeln et al., 1998; Soderstrom et al., 2003) severely undercuts the Smith et al. argument.

A more direct way to assess common causes is to determine whether measures of putative common causes, when introduced into statistical prediction equations, explain observed associations between smoking and suicidal behaviors. A number of epidemiological studies have done this. In the most detailed analysis of this sort to date, Hemmingsson and Kriebel (2003) studied the prospective association between baseline smoking and suicide over a 26-year follow-up period in a sample of nearly 50,000 Swedish men initially surveyed in conjunction with compulsory military conscription. A 3-fold elevated risk of suicide was found in the Swedish death registry among baseline heavy smokers versus nonsmokers. This elevated risk almost entirely disappeared, though, when variables were introduced into the prediction equations for baseline risk factors that included alcohol consumption, emotional control, parental divorce, contact with the police, use of medication for nervous problems, and a follow-up measure of later contact with the health care system for alcoholism.

A number of other prospective epidemiological studies have examined the extent to which independent risk factors explain the association between smoking and suicidal behaviors. Included here are prospective studies of suicide in general population samples (Leistikow et al., 2000; Miller et al., 2000a,b; Tanskanen et al., 2000), cross-sectional and longitudinal studies of suicidal ideation, plans, and attempts in general population samples (Hallfors et al., 2004; Hintikka et al., 2001; King et al., 2001; Wu et al., 2004), and studies of suicidal behaviors in psychiatric treatment samples (Makikyro et al., 2004; Oquendo et al., 2004; Potkin et al., 2003). Although the smoking-suicidality association always attenuated in these studies when measures of risk factors were controlled, the association always remained statistically significant. However, as the risk factor measures in these studies were generally much less complete than in the Hemmingsson and Kriebel study, a question can be raised whether the smoking-suicidality association would have disappeared with more complete controls.

The current report presents the first results from a nationally representative US survey on whether the association between smoking and suicidal behaviors can be explained by controlling for a comprehensive set of mental disorders. The data come from the National Comorbidity Survey-Replication (NCS-R) (Kessler and Merikangas, 2004), a nationally representative survey of the US household population. Although the analysis is based only on cross-sectional data rather than on longitudinal data, and the outcomes are measures of suicide-related behaviors (suicide ideation, plans, and attempts) rather than completed suicides, documentation that strong associations of smoking with the outcomes can be explained with comprehensive controls for measures of mental illness would be of considerable value.

Based on the fact that a number of previous epidemiological studies have documented dose–response relationships between smoking and suicidality that involve either intensity of smoking (e.g., number of cigarettes smoked each day) (Hemmingsson and Kriebel, 2003) or recency of smoking (i.e., current versus past smokers) (Miller et al., 2000b), we began by building multivariate models of the dose–response relationships between smoking and the 3 suicidal behavior outcomes. We then evaluated whether controlling the DSM-IV mental disorders explains the associations between smoking and these outcomes.

METHODS AND MATERIALS

Sample

The NCS-R is a psychiatric epidemiological survey carried out in a multistage clustered area probability sample of the US household population between February 2001 and April 2003. The survey was administered by the interview staff of the Survey Research Center at the University of Michigan. The sample was restricted to English-speaking household residents ages 18 and over (Kessler and Merikangas, 2004). Face-to-face interviews were completed in the homes of 9282 respondents. The response rate was 70.9%. Sample recruitment began with an advance letter and Study Fact Brochure to introduce the study. Interviewers then followed up with in-person visits to answer questions before obtaining verbal informed consent. Consent was verbal rather than written to mimic the procedures used in the baseline NCS survey (Kessler et al., 1994) for purposes of trend comparison. Respondents were given $50 for participation. A probability subsample of initial nonrespondents was asked to complete a brief telephone nonrespondent survey to check for nonresponse bias. A $100 incentive was offered for participation.

The Human Subjects Committees of Harvard Medical School and the University of Michigan both approved these recruitment and consent procedures.

The NCS-R interview was administered in 2 parts. Part I included a core diagnostic assessment of DSM-IV mental disorders. Part II included questions about correlates and additional disorders. Part II was administered to a probability subsample of 5692 Part I respondents that included 100% of those who met lifetime criteria for any Part I disorder and a probability subsample of others. Smoking was assessed in Part II. The Part II sample was weighted to adjust for differential probabilities of selection, over-sampling of Part I respondents with a mental disorder, nonresponse, and residual differences between the sample and the year 2000 Census on sociodemographic and geographic variables. A more detailed description of NCS-R weighting is presented elsewhere (Kessler et al., 2004).

Suicidal Behavior

The NCS-R questions about suicidal behaviors followed those in the baseline NCS (Borges et al., 2000; Kessler et al., 1999) in asking about lifetime occurrence, age of onset, and recency of suicidal ideation ("Have you ever seriously thought about committing suicide?"), suicide plans ("Have you ever made a plan for committing suicide?"), and suicide attempts ("Have you ever attempted suicide?"). The outcomes considered here are past year occurrence of these outcomes, as we were interested in examining dose–response relationships with quantity-frequency of smoking assessed for smoking in the year before interview.

Smoking

Respondents were asked if they ever smoked even a single puff of a cigarette, cigar, or pipe, and, if so, the age when they took their first puff. Parallel lifetime and age of onset questions were then asked about smoking at least once per week for 2 months or longer and smoking every day or nearly every day for 2 months or longer. Respondents who ever smoked regularly were then asked about number of days smoked in the past 12 months and the average number of cigarettes, cigars, and pipes smoked per day in the past 12 months. These questions were then repeated for the year of heaviest smoking. Information was then collected on number of years of daily smoking followed by the assessment of lifetime DSM-IV nicotine dependence (as shown in the next section) and the Fagerstrom Test for Nicotine Dependence (FTND) (Heatherton et al., 1991). The FTND distinguishes within the subset of DSM-IV dependence cases those with a physiological dependence syndrome (Hughes et al., 2004; Moolchan et al., 2002).

DSM-IV Mental Disorders

DSM-IV mental disorders were included as control variables to determine whether they statistically explain any gross associations between smoking and suicidal behaviors. The mental disorders and nicotine dependence were assessed using Version 3.0 of the WHO Composite International Diagnostic Interview (CIDI) (Kessler and Ustun, 2004), a fully structured lay-administered diagnostic interview. Both lifetime and past year disorders were assessed. The 18 disorders considered here included anxiety disorders (panic disorder, generalized anxiety disorder, agoraphobia, specific phobia, social phobia, obsessive-compulsive disorder, posttraumatic stress disorder), mood disorders (major depressive disorder, bipolar I and II disorders, dysthymic disorder), substance use disorders other than nicotine dependence (alcohol and drug abuse with and without dependence), and impulse-control disorders (oppositional-defiant disorder, conduct disorder, attention-deficit/hyperactivity disorder, intermittent explosive disorder). Organic exclusion rules and diagnostic hierarchy rules were used in making diagnoses. As detailed elsewhere (Kessler et al., 2005), fair-good concordance was found between CIDI diagnoses and clinical diagnoses based on the Structured Clinical Interview for DSM-IV (SCID) (First et al., 2002), with area under the receiver operating characteristic curve .65–.81 for anxiety disorders, .75 for major depressive episode, .62–.88 for substance disorders, and .76 for any anxiety, mood, or substance disorder. No validation was made of the impulse-control disorders, as these are not assessed in the SCID.

Sociodemographic Controls

All logistic regression analyses included controls for age (18–29, 30–44, 45–59, 60+), sex, race–ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, other), marital status (married, separated, divorced, widowed, never married; and a distinction between cohabiting and not cohabiting for each of the categories other than married), employment status (employed or self-employed, student, homemaker, retired, and other), and family income (less than 1.5, 1.5–3, greater than 3–6, and greater than 6 times the federal poverty line for a family the size and composition of the respondent's family). The aim in using these controls was to adjust for the effects of ascribed (i.e., age, sex, race–ethnicity) and achieved (i.e., marital status, employment status) social statuses and socioeconomic status (indicated by family income) that could be associated both with different norms regarding smoking and with differential risk for stressful experiences that predispose to suicidal behaviors.

Analysis Methods

Logistic regression analysis was used to fit models for gross associations of smoking in predicting 12-month suicidal behaviors with controls for sociodemographics. Three dichotomous outcomes were examined: (ai) suicide ideation (with or without a plan or attempt) versus no suicide ideation or plan or attempt; (b) suicide plan (with or without an attempt) versus no suicide plan or attempt (with or without ideation); and (c) suicide attempt (with or without a plan) versus no suicide attempt (with or without ideation or plan). These 3 outcomes are not independent. It would have been possible to define independent outcomes (e.g., attempt among planners), but this would have divided the small number of respondents with suicidal behaviors into even smaller subsets, yielding less stable results than those obtained here. As our goal was to test whether meaningful net associations exist between smoking and these outcomes rather than to investigate causal pathways by which smoking causes attempts through other

outcomes, we opted for the aggregated approach to define outcomes.

In carrying out the analyses, we created successively more complex measures of smoking as independent variables in logistic regression equations to predict 12-month suicidal behaviors. We began with 2 dichotomous measures of current and past lifetime smoking, then elaborated each of these 2 to distinguish 3 classes of smoking frequency: occasional (less than once per week), regular (at least once per week, but less than daily), and daily. We next refined the category of daily use to distinguish quantity of cigarette smoking (half a pack or less, between a half and a full pack, more than 1 pack per day) and history of daily use (1–10 years, 11–20 years, and 21+ years) in the subsample of smokers who smoked cigarettes. The numbers of cigar and pipe smokers were too few to allow powerful analyses to be carried out of quantity of use, although these individuals were combined with cigarette smokers in the analysis of frequency of use. Finally, information was added to the prediction equations about lifetime and 12-month DSM-IV nicotine dependence and the FTND severity categories of 12-month physiological dependence (low or very low, medium, high or very high). All significant predictors were included in the successively more refined equations. Our purpose in working with this complex set of smoking measures was to determine whether dose–response relationships could be documented with suicidal behaviors. Although use of so many smoking measures might lead to capitalizing on chance in finding associations judged to be statistically significant, we wanted to be exhaustive in developing the strongest possible prediction equation before attempting to explain away these associations with controls for mental disorders.

Once final models were developed for the associations of smoking with suicidal behaviors, controls were introduced for 12-month DSM-IV/CIDI disorders. Although 18 mental disorders were assessed, we did not want to include this large number of controls for fear of over-fitting the data. Four summary count variables were consequently created, one for the number of disorders the respondent had in each of the 4 broad categories of anxiety disorders (0–7), mood disorders (0–2), substance use disorders other than nicotine dependence (0–4), and impulse-control disorders (0–4). The significance of these 4 variables was evaluated globally using Wald χ^2 tests. The coefficients of interest were those associated with smoking before and after introducing the measures of mental disorders. Coefficients were exponentiated and are presented as odds ratios (ORs). We also evaluated statistical interactions between smoking and mental disorders using Wald χ^2 tests. Standard errors of estimates were obtained using the Taylor series linearization method (Wolter, 1985) implemented in the SAS software system (SAS Institute Inc., 2002) to adjust for the weighting and clustering of the NCS-R data. The Wald χ^2 tests used design-corrected coefficient variance-covariance matrices. Statistical significance was evaluated using 2-sided design-based .05 level tests.

RESULTS

Smoking and Suicidal Behaviors

Twelve-month prevalence (standard error and unweighted n in parentheses) of suicidal behaviors in the NCS-R are 2.6% (0.2; $n = 229$) for ideation (with or without a plan or attempt), 0.7% (0.1; $n = 68$) for suicide plans (with or without an attempt), and 0.5% (0.1; $n = 49$) for suicide attempts. The ORs of current smoking versus never smoking are moderately elevated (i.e., greater than 1.0) and statistically significant in predicting 2 of these 3 outcomes—thoughts (1.8) and attempts (3.2)—and elevated but not statistically significant in predicting suicide plans (1.5) in a basic model (Model I) that controls for sociodemographic variables (Table 1). (Coefficients for control variables are not shown, but are available on request.) The ORs for past smoking are not meaningfully different from 1.0 in these 3 equations (0.3–1.1). Elaboration of the basic model (Model II) shows that even though current smoking is a predictor of thoughts and attempts, neither frequency of current use (i.e., the number of days in a typical month the respondent smokes) ($\chi^2_2 = 0.7$–1.6, $p = .45$–$.79$) or highest lifetime cigarette smoking frequency ($\chi^2_2 = 1.2$–3.6, $p = .16$–$.55$) is meaningfully related to any outcome among past cigarette smokers. A more elaborate model that included a continuous variable for number of years of use with heaviness of use failed to add significantly to prediction accuracy.

Further elaboration of the basic model (Model III) documented a strong dose–response relationship between number of cigarettes smoked per day and ideation among current daily cigarette smokers ($\chi^2_2 = 12.2$, $p = .002$) and a trend relationship of a similar sort predicting suicide plans ($\chi^2_2 = 4.6$, $p = .10$). The ORs associated with heavy cigarette smoking in these equations (3.4–3.5) are more than twice those associated with light cigarette smoking (1.2–1.4), with the ORs of moderate cigarette smoking intermediate between these extremes (1.7–2.1). Quantity of current cigarette smoking is not meaningfully related to attempts ($\chi^2_2 = 0.2$, $p = .89$). Nor is highest lifetime quantity of cigarette use meaningfully related to ideation among past cigarette smokers ($\chi^2_2 = 0.2$, $p = .91$). Model convergence could not be achieved in predicting plans or attempts from past smoking frequency due to sparseness of data. More complex models (results not shown, but available on request) were also estimated to investigate effects of smoking history. None of these predictors was significantly related to any of the outcomes, including age at onset of smoking ($\chi^2_2 = 0.2$–1.2, $p = .925$–$.537$), number of lifetime years smoking ($\chi^2_2 = 0.9$–1.7, $p = .43$–$.63$), and number of lifetime attempts to quit ($\chi^2_2 = 0.8$–3.2, $p = .20$–$.67$).

DSM-IV Nicotine Dependence and Suicidal Behaviors

DSM-IV nicotine dependence is not meaningfully related to the outcomes after controlling for quantity-frequency of smoking ($\chi^2_2 = 0.6$–2.9, $p = .23$–$.74$) (Table 2). The FTND measure of 12-month physiological dependence is a strong predictor of any of the outcomes either among respondents who meet criteria for 12-month DSM-IV nicotine

TABLE 1. The Associations (Odds Ratios) of Smoking With 12-mo Suicidal Ideation, Plan, and Attempt as a Function of Smoking Recency, Frequency, and Quantity Among Part II NCS-R Respondents ($n = 5692$)[a]

	Ideation				Plan		Attempt	
	%[b]	*SE*	OR	95% CI	OR	95% CI	OR	95% CI
Model I (recency)								
Current smoking	29.6	0.8	1.8*	1.2–2.6	1.5	0.7–3.0	3.2*	1.6–6.5
Past smoking	20.1	0.9	0.9	0.4–1.9	0.3	0.1–1.0	1.1	0.2–6.7
χ^2_2	—	—		11.8*		10.6*		10.9*
Model II (frequency)[c]								
Current smoking								
Occasional	10.4	1.0	1.6	0.7–3.6	1.1	0.2–5.4	1.9	0.4–9.1
Regular	13.1	1.1	1.3	0.6–2.5	1.3	0.5–3.5	4.1*	1.2–14.0
Daily	76.4	1.4	1.9*	1.3–2.8	1.6	0.8–3.2	3.4*	1.7–6.8
χ^2_2	—	—		1.6		0.5		0.7
Past smoking								
Occasional	7.3	1.2	0.2	0.0–1.6	0.5	0.1–4.3	—[d]	—
Regular	5.9	0.8	0.3	0.0–1.8	0.8	0.1–5.9	—	—
Daily	86.8	1.5	1.0	0.5–2.3	0.2	0.0–1.0	—	—
χ^2_2	—	—		3.6		1.2		—
Model III (quantity)								
Current smokers[c]								
Light (1–10)	53.1	1.9	1.2	0.7–2.0	1.4	0.5–3.4	3.2	1.4–7.2
Moderate (11–20)	34.2	1.7	2.1*	1.2–3.5	1.7	0.7–4.1	3.3*	1.4–7.7
Heavy (21+)	12.9	1.1	3.4*	1.8–6.3	3.5*	1.5–8.2	2.4	0.9–6.5
χ^2_2	—	—		12.2*		4.6		0.2
Past smokers[c]								
Light (1–10)	44.1	2.5	1.0	0.3–3.2	—[d]	—	—[d]	—
Moderate (11–20)	27.4	1.3	1.2	0.6–2.7	—	—	—	—
Heavy (21+)	29.2	2.3	1.0	0.4–2.8	—	—	—	—
χ^2_2	—	—		0.2		—		—

*Significant at the .05 level, 2-sided test.
[a]Controlling for age, sex, race–ethnicity, occupational status, marital status, family income.
[b]Prevalence of predictors in the total sample (I) or among current smokers and past smokers (II–III).
[c]Frequency was defined in terms of number of days per month the respondent smoked, while quantity was defined in terms of number of smokes per day.
[d]Associations could not be estimated due to sparse data.

dependence ($\chi^2_2 = 0.0–4.1$, $p = .13–.88$) or among respondents with a history of past dependence ($\chi^2_2 = 0.2–4.8$, $p = .09–.90$). However, high FTND scores do significantly predict suicidal ideation among respondents with both current and past DSM-IV nicotine dependence. These ORs are virtually identical (2.4–2.4), which means that recency of dependence is less important than dependence being present in the life course. A broadly similar pattern was found in predicting plans and attempts, with the ORs of FTND scores consistently elevated and comparable for current (1.6–2.8) and past (1.7–2.9) dependence.

Comparison of Gross and Net Effects of Smoking Controlling DSM-IV Disorders

The smoking measures are consistently and significantly related to 12-month DSM-IV/CIDI mental disorders, with ORs higher for measures of substance use disorders (median OR of 5.1, with an interquartile range of 3.1–7.6) than anxiety (2.0, 1.8–2.7), mood (2.5, 1.8–3.1), or impulse-control (2.2, 2.0–3.6) disorders (Table 3). The ORs involving each mental disorder are generally higher with the 2 indicators of nicotine dependence (3.5, 2.3–43) than with measures of smoking (1.8, 1.7–2.8) or quantity-frequency of smoking (2.2, 1.8–25). Consistent with findings in the baseline NCS (Kessler et al., 1999), mental disorders are also consistently related to the 3 measures of suicidality (results not shown, but available on request). Based on these results, it is plausible to think that statistical control for mental disorders would reduce the associations between smoking and suicidal behaviors.

We tested the possibility that controls for mental disorders explain the associations of smoking with suicidal behaviors by developing a separate multivariate model for the gross effects of the smoking variables in predicting each suicidality outcome. We then estimated a net effects model that added controls for the 4 summary measures of 12-month mental disorders. The smoking variables differed somewhat across the outcomes to maximize strength of association between smoking and suicidal behaviors (see notes in Table 4

TABLE 2. The Associations (Odds Ratios) of DSM-IV Nicotine Dependence of 12-mo Suicidal Ideation, Plans, and Attempts Among Part II NCS-R Respondents (n = 5692)[a]

	Ideation				Plan		Attempt	
	%[b]	*SE*	OR	95% CI	OR	95% CI	OR	95% CI
Model IV (DSM-IV nicotine dependence)								
Current	4.0	0.3	1.6	0.9–2.7	2.2	0.9–5.3	1.4	0.6–3.5
Past	4.2	0.3	1.0	0.5–2.4	1.6	0.5–5.7	1.4	0.2–9.3
χ^2_2	—	—		2.9		4.4		0.6
Model V (FTND scores among respondents with current and past DSM-IV nicotine dependence)[c]								
Current dependence								
Low	53.0	4.2	1.2	0.7–2.2	2.5	0.8–8.1	1.2	0.3–4.5
Moderate	14.4	3.6	1.1	0.3–4.2	—	—	1.6	0.2–10.3
High	32.6	4.2	2.4*	1.2–4.8	2.8	1.0–7.9	1.6	0.4–6.4
χ^2_2	—	—		4.1		0.0		0.1
Past dependence								
Low	100.0	—	1.1	0.7–1.8	2.1	0.9–4.6	1.3	0.4–3.9
Moderate	0.0	—	1.1	0.3–4.2	—	—	1.6	0.2–12.0
High	0.0	—	2.4*	1.2–4.9	2.9*	1.1–8.0	1.7	0.4–6.5
χ^2_2	—	—		4.8		0.3		0.0.2

*Significant at the .05 level, 2-sided test.

[a]Controlling for age, sex, race–ethnicity, occupational status, marital status, and family income and for recency, frequency, and quantity of smoking.

[b]Prevalence of predictors in the total sample (I) or among respondents with current and past DSM-IV nicotine dependence (II).

[c]Fagerstrom screening test for nicotine dependence (FTND).

for details). In all 3 cases, the predictors include a measure of quantity or frequency or combined quantity/frequency of current smoking as well as a term for either DSM-IV nicotine dependence or the FTND measure of physiological dependence. The overall gross associations of these smoking variables are statistically significant in predicting all 3 outcomes (χ^2_{3-4} = 12.7–36.6, p = <.001–.003), with a consistent dose–response relationship between smoking quantity and/or frequency and each outcome and ORs in the range 2.9–3.0 for the highest values of the predictors. (Table 4) The dependence measure has a consistently elevated OR in the range 1.4–2.3.

The associations of the smoking variables are no longer significant in the net effects model (χ^2_{3-4} = 2.4–5.1, p = .28–.67) after controlling for mental disorders. Nor is any of the individual smoking variables either significant in statistical terms or substantially elevated (i.e., ORs of 2.0 or greater) in this model. The 4 summary mental disorder count variables, in comparison, are significant in predicting all 3 outcomes (χ^2_4 = 164.5–357.0, p < .001). An exploratory analysis failed to find significant interactions between number of DSM-IV disorders and the smoking variables (χ^2_{3-4} = 1.4–6.6., p = .16–.70). Inspection of results in additive models that controlled only mental disorders of one type (results not shown, but available on request) shows that mood disorders are most important for suicidal ideation and substance use disorders most important for suicide plans and attempts, although all 4 classes of mental disorders are associated with important reductions in the smoking ORs predicting all 3 outcomes.

DISCUSSION

These results should be interpreted in light of 5 limitations: First, the prevalence of suicidal behaviors is low, limiting statistical power. Second, analyses were based on cross-sectional naturalistic data. Neither temporal nor causal priority can be established even though we treated measures of smoking as the predictors and suicidal behaviors as the outcomes. Third, the assessment of smoking excluded consideration of smokeless tobacco use, limiting the generality of results. Fourth, the assessment of mental disorders was based on self-reports rather than clinical assessments. Although blinded clinical reappraisal interviews found good concordance between CIDI diagnoses and clinical diagnoses, some respondents might have consciously and consistently failed to disclose information about mental disorders and suicidality in both the main survey and the clinical reappraisal interviews. Fifth, the ad hoc model fitting for the smoking variables might have resulted in overestimating the strength of the associations between smoking and the outcomes. As noted above, though, this was done consciously to investigate dose–response relationships and to create a strong baseline model to evaluate the effects of controlling for mental disorders.

Within the context of these limitations, the documentation of elevated gross cross-sectional ORs between recent smoking and recent suicidal behaviors is consistent with other cross-sectional surveys (Brener et al., 1999; Frank and Dingle, 1999; Hallfors et al., 2004; King et al., 2001). The finding that current smoking is a more powerful predictor than past smoking is

TABLE 3. The Associations (Odds Ratios) of 12-mo Smoking Quantity, Frequency, and Dependence With 12-mo DSM-IV/CIDI Disorders Among Part II NCS-R Respondents (n = 5692)[a]

	Current Smoking		Daily Smoking		Heavy Daily Smoking		12-mo DSM-IV Nicotine Dependence		High FTND Score[b]	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Mood disorders										
Major depressive disorder	1.8*	1.5–2.1	1.8*	1.5–2.2	2.4*	1.7–3.3	3.0*	2.2–4.1	4.5*	2.8–7.1
Bipolar disorder	2.8*	2.0–3.9	2.6*	1.9–3.6	2.5*	1.6–4.0	3.1*	2.0–4.8	4.1*	2.2–7.8
Dysthymia	1.7*	1.2–2.4	1.8*	1.3–2.5	2.2*	1.3–3.7	3.0*	1.7–5.1	4.3*	2.7–6.9
Anxiety disorders										
Panic disorder	1.9*	1.4–2.6	1.9*	1.4–2.4	1.5	0.8–2.6	2.3*	1.3–4.2	2.1*	1.1–4.2
Generalized anxiety disorder	1.7*	1.3–2.1	1.6*	1.2–2.1	2.4*	1.5–3.9	2.9*	2.0–4.4	3.9*	2.4–6.4
Agoraphobia	1.6*	1.0–2.7	1.9*	1.2–2.9	1.9*	1.0–3.4	1.8*	1.0–3.4	1.6	0.6–3.8
Social phobia	1.4*	1.1–1.7	1.5*	1.2–1.8	1.8*	1.2–2.7	2.4*	1.6–3.7	3.5*	2.1–5.9
Specific phobia	1.4*	1.1–1.8	1.6*	1.3–2.0	2.1*	1.6–2.7	2.7*	2.0–3.6	2.9*	2.0–4.3
Posttraumatic stress disorder	1.8*	1.4–2.2	1.8*	1.4–2.4	2.6*	1.6–4.0	3.8*	2.4–5.9	4.4*	2.4–7.8
Obsessive compulsive disorder	2.0	0.8–5.0	2.5	0.9–6.4	2.0	0.6–6.8	2.7*	1.0–7.5	4.7*	1.6–3.5
Impulse-control disorders										
Intermittent explosive disorder	2.1*	1.6–2.8	2.0*	1.6–2.5	2.3*	1.4–3.7	2.3*	1.3–3.9	3.8*	2.1–6.8
Oppositional-defiant disorder	2.1*	1.1–3.9	2.1*	1.1–4.0	2.6*	1.2–5.7	2.1	0.9–4.7	4.6*	1.3–15.9
Conduct disorder	1.8	0.7–5.0	1.9	0.8–4.7	2.6	0.8–8.2	3.6*	1.4–8.9	2.2	0.4–11.5
Attention deficit/hyperactivity	1.8*	1.2–2.6	1.9*	1.4–2.8	3.2*	1.8–5.8	4.2*	2.6–7.0	5.3*	2.9–9.8
Substance use disorders										
Alcohol abuse or dependence	5.8*	3.8–8.7	4.2*	3.0–5.8	2.2*	1.3–3.9	2.7*	1.5–4.9	2.9*	1.4–5.8
Alcohol dependence	7.6*	4.1–14.3	4.6*	2.8–7.5	4.3*	2.5–7.5	5.0*	2.5–9.8	5.1*	2.0–12.9
Illicit drug abuse or dependence	7.2*	3.8–13.9	5.7*	3.0–10.5	3.1*	1.5–6.6	6.5*	3.8–11.1	8.3*	3.4–20.4
Illicit drug dependence	10.2*	4.3–23.9	7.0*	3.2–15.6	1.6	0.3–8.1	8.3*	3.3–20.6	9.1*	3.1–27.0

DSM-IV indicates diagnostic and statistical manual of mental disorders, fourth edition; NCS-R, National Comorbidity Survey Replication.

*Significant at the .05 level, 2-sided test.

[a]Odds ratios were estimated in separate logistic regression equations with only one smoking variable predicting each mental disorder controlling for age, sex, race–ethnicity, occupational status, marital status, and family income. Smoking variables with more than one category were dichotomized and their highest category compared to all other categories in the total sample (e.g., heavy daily smoking versus all other respondents whether they are less than heavy daily smokers, smokers that smoke less than daily, or nonsmokers).

[b]Fagerstrom screening test for nicotine dependence (FTND).

consistent with the small number of previous studies that examined that specification (King et al., 2001; Miller et al., 2000b). The finding of a dose–response relationship between quantity of current smoking and suicidal ideation is consistent with the 2 previous studies that examined that association (King et al., 2001; Wu et al., 2004).

The specifications involving quantity, frequency, history, and recency of dependence go beyond previous research and generally are inconsistent with most plausible hypotheses that implicate smoking as a cause of suicidal behaviors because they do not document a dose–response relationship between smoking and suicidal behaviors. The most important of these specifications are that quantity of cigarette smoking is not related either to suicide plans or attempts, that the ORs for nicotine dependence are very similar for remitted and current cases, and that number of years smoking is unrelated to the outcomes either among current or past smokers.

As noted in the introduction, previous studies have generally failed to explain the associations between smoking and suicidal behavior outcomes with the less detailed measures of mental disorders included in their analyses. We found, in comparison, that these associations can be explained with controls for the more carefully and comprehensively assessed CIDI diagnoses. Although it is not possible to make a clear adjudication between contending causal hypotheses to explain the mechanism involved in this finding, the finding makes it clear that future research on smoking and suicidal behavior needs to focus much more centrally than most previous research on mental disorders either as common causes, markers, or mediators.

Our findings are consistent both with the common cause hypothesis (i.e., that both current smoking and current suicidal behaviors are consequences either of mental and substance use disorders or of more distal causes for which current mental and substance use disorders serve as proxies) and with the mediation hypothesis (i.e., that smoking has a causal effect on suicidal behaviors that is mediated by mental disorders), although absence of a dose–response relationship between quantity-frequency of smoking and the suicidal behavior argues indirectly against the mediation hypothesis. Prospective cohort studies are needed to resolve the problem of distinguishing between common causes and mediation. This can happen, though, only when a clear temporal order exists between the putative common cause (e.g., mental disorder) and the independent variable (e.g., smoking). It is important to note that this temporal order has never been

TABLE 4. Associations (Odds Ratios) of 12-mo Smoking Quantity, Frequency, and Dependence With 12-mo Suicidal Ideation, Plan, and Attempt in Gross Effects Models (i.e., Models With Controls Only for Sociodemographic Variables) and Net Effects Models (i.e., Models That Also Controlled for DSM-IV/CIDI Mental Disorders) Among Part II NCS-R Respondents ($n = 5692$)

	Ideation				Plan				Attempt			
	Gross Effects[a]		Net Effects[a]		Gross Effects[a]		Net Effects[a]		Gross Effects[a]		Net Effects[a]	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Current quantity-frequency of smoking												
Low[b]	1.3	0.8–1.9	1.0	0.6–1.5	1.2	0.5–2.8	1.0	0.5–2.1	1.8	0.4–8.6	1.2	0.2–6.3
Intermediate[b]	1.9*	1.1–3.2	1.3	0.7–2.3	1.4	0.6–3.4	0.9	0.3–2.6	—[c]	—	—	—
High[b]	3.0*	1.6–5.4	1.8	0.9–3.6	2.9*	1.3–6.3	1.9	0.8–4.5	3.1*	1.4–6.8	2.2	0.8–6.1
χ^2_3	17.6*		4.1		8.8*		2.5		$\chi^2_2 = 8.1$*		2.6	
Nicotine dependence												
High FTND[e]	2.3*	1.2–4.6	1.2	0.6–2.8	—[c]	—	—	—	—[c]	—	—	—
DSM-IV[d]	—[c]	—	—	—	2.0*	1.1–3.8	0.9	0.4–2.0	1.4	0.5–3.7	0.6	0.2–1.4
χ^2_1	5.9*		0.3		4.4*		0.0		0.5		1.5	

*Significant at the .05 level, two-sided test.

[a]The gross effects models control for age, sex, race–ethnicity, occupational status, marital status, and family income. The net effects models control for the same sociodemographic variables plus 4 summary measures of 12-mo DSM-IV/CIDI mental disorders. The summary measures are counts of the numbers of mood, anxiety, impulse-control, and substance use disorders for which the respondent meets 12-mo diagnostic criteria. Coefficients for the control variables are not shown, but are available on request. The mental disorders had significant effects as a set in predicting each of the 3 outcomes in the net effects models ($\chi^2_4 = 164.5–357.0$, $p < .001$).

[b]The definitions of low, intermediate, and high smoking quantity-frequency differ across outcomes because the predictors were selected to optimize strength of associations with individual outcomes based on the results of preliminary analyses. Information about past quantity-frequency of smoking was not included in predicting any of the outcomes based on the finding of nonsignificant associations in preliminary analyses. For suicidal ideation, low quantity-frequency included all occasional and regular smokers plus the subset of daily cigarette smokers who smoke 1–10 cigarettes per day. Intermediate included daily cigarette smokers who smoke 11–20 cigarettes per day. High included daily cigarette smokers who smoke 21+ cigarettes per day. For suicide plans, low included all regular smokers plus the subset of daily cigarette smokers who smoke 1–10 cigarettes per day. (Note that occasional smokers were excluded and combined with non-smokers in the contrast group.) Intermediate- and high quantity-frequency was defined as in the models for suicidal ideation. For suicide attempts, low included all current occasional smokers, while high included all current regular and daily smokers. Smokers who exclusively smoke cigars and/or pipes were classified low in predicting all outcomes. Sensitivity analysis showed that results are not affected by modifying this last coding rule to classify exclusive cigar-pipe smokers as moderate or high.

[c]This smoking measure was not included in the final model for this outcome because it was found not to be significant in preliminary analyses.

[d]DSM-IV nicotine dependence was defined as present among both past and current cases. This means that respondents who no longer smoked were coded yes on this predictor if they were dependent at some time in their life. The results in Table 2 show that this combination of past with current cases is justified empirically by the strong similarity in ORs across the 2 subsamples.

[e]High FTND was defined as a high or very high score on the Fagerstrom screening Test for Nicotine Dependence (FTND) using published cut-points among respondents with a lifetime history of DSM-IV nicotine dependence whether past or current. The results in Table 2 show that this combination of past with current cases is justified empirically by the strong similarity in ORs across the 2 subsamples.

established in prospective cohort studies of smoking and suicidal behavior. In the Hemmingsson and Kriebel 26-year prospective study briefly described in the introduction, for example, smoking and emotional problems were both measured at baseline, making it impossible to distinguish between a temporal order in which early smoking predicted the subsequent onset of emotional problems that were already in existence as of the baseline assessment, the reverse, or a situation where unmeasured common causes led to both.

Future research needs to be designed in such a way as to distinguish temporal and causal priorities between smoking and mental disorders to resolve this uncertainty. Although based on retrospective data, previously reported analyses in the baseline NCS using retrospective age-of-onset reports illustrate the logic required in future prospective investigations. Those earlier analyses found reciprocal predictive associations of (a) temporally primary regular smoking with subsequent onset of mental disorders (Breslau et al., 2004a) and (b) temporally primary mental disorders with subsequent onset of regular smoking (Breslau et al., 2004b). The evidence for mental disorders predicting subsequent onset of smoking was much stronger, though, than of smoking predicting subsequent onset of mental disorders. This finding is consistent with the interpretation that mental disorders, or some causal factors of which mental disorders are markers, cause both smoking and suicidal behaviors. The ultimate test of this hypothesis, of course, would require an intervention that manipulated current smoking and determined whether this resulted in a reduction in suicide. Although such an experiment would probably be logistically intractable, a practical alternative might be a quasi-experimental evaluation that took advantage of aggregate time-space variation in some policy variable (e.g., size of cigarette tax) that affected smoking behavior for reasons that could plausibly be assumed to be otherwise independent of suicidal behaviors.

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